RISK FACTORS OF BIRTH ASPHYXIA

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Background: Birth asphyxia is a serious clinical problem worldwide. There are many reasons a baby may not be able to take in enough oxygen before, during, or just after birth. Damage to brain tissues is a serious complication of low oxygen that can cause seizures and other neurological problems. This study was designed to asses the risk factors of birth asphyxia in neonates. **Methods:** This descriptive, prospective study was conducted in the Department of Paediatrics, Isra University Hospital, Hyderabad, from April 2005 to April 2006. 125 newborn (75 males and 50 females) admitted to the neonatal care unit, who were delivered with delayed cry or low apgar score (<7) were included. Detailed maternal history was taken, regarding their age, gestational age, and complications, if any. **Results:** Out of 125 neonatal encephalopathy cases, 28% were diagnosed as suffering with moderate or severe encephalopathy, whereas 36% had mild encephalopathy. Risk of neonatal encephalopathy increased with increasing or decreasing maternal age. Antepartum risk factors included non-attendance for antenatal care (64%). Multiple births increased risk in 4.8%. Intrapartum risk factors included non-cephalic presentation (20%), prolonged rupture of membranes (24%) and various other complications. Particulate meconium was associated with encephalopathy in 9.6%. 60% mothers were anemic. Vaginal bleeding was strongly associated with birth asphyxia in 34.44% of neonates. 56% of mothers delivered at home, while 28% delivered at a private hospital or maternity home. Only 12% delivered at a tertiary care hospital. **Conclusion:** Lack of antenatal care, poor nutritional status, antepartum hemorrhage and maternal toxaemia were associated with higher incidence of asphyxia. Improvements in the public health of women with associated gains in female growth and nutrition must remain a longer-term goal. Early identification of high-risk cases with improved antenatal and perinatal care can decrease such high mortality. Safe motherhood policy is recommended.

Key words: Risk factor; Birth asphyxia; Hypoxic ischemic encephalopathy (HIE)

INTRODUCTION

Birth asphyxia is a serious clinical problem worldwide. Each year approximately 4 million babies are born asphyxiated, which results in 1 million deaths and an equal number of serious neurological sequelae, such as cerebral palsy, mental retardation, and epilepsy [1]. Asphyxia is an insult to the fetus or newborn due to lack of oxygen or lack of perfusion to various organs [2]. There are many reasons a baby may not be able to take in enough oxygen before, during, or just after birth. A mother may have medical conditions that can lower her oxygen levels; there may be a problem with the placenta that prevents enough oxygen from circulating to the fetus; or the baby may be unable to breath after delivery.

Damage to the brain tissue is a serious complication of low oxygen that can cause seizures and other neurological problems.[3-5] Clinical manifestations and course vary depending on Hypoxic Ischemic Encephalopathy (HIE) severity. In mild HIE, muscle tone may be increased slightly and deep tendon reflexes may be brisk during the first few days. Transient behavioral abnormalities, such as poor feeding, irritability, or excessive crying or sleepiness, may be observed. By 3-4 days of life, the central nervous system examination findings become normal. In moderately severe HIE, the infant is lethargic, with significant hypotonia and diminished deep tendon reflexes. The grasping, Moro, and sucking reflexes may be sluggish or absent. The infant may experience occasional periods of apnea. Seizures may occur within the first 24 hours of life. Full recovery within 1-2 weeks is possible and is associated with a better long-term outcome. An initial period of well being or mild HIE may be followed by sudden deterioration, suggesting ongoing brain cell dysfunction, injury, and death; during this period, seizure intensity might increase. In severe HIE, stupor or coma is typical. The infant may not respond to any physical stimulus. Breathing may be irregular, and the infant often requires ventilatory support. Generalized hypotonia and depressed deep tendon reflexes are common. Neonatal reflexes (e.g sucking, swallowing, grasping, Moro) are absent. Disturbances of ocular motion may be present. Pupils may be dilated, fixed, or poorly reactive to light. Seizures occur early and often and may be initially resistant to conventional treatments. The seizures are usually generalized, and their frequency may increase during the 24-48 hours after onset, correlating with

the phase of reperfusion injury. As the injury progresses, seizures subside and the electroencephalogram becomes isoelectric or shows a burst suppression pattern. At that time, wakefulness may deteriorate further, and the fontanel may bulge, suggesting increasing cerebral edema. Irregularities of heart rate and blood pressure are common during the period of reperfusion injury, as is death from cardio respiratory failure.[6,7] The burden of perinatal mortality and morbidity falls disproportionately on low-income populations.

The World Health Organization (WHO) estimates that the overall perinatal mortality rate in low-income countries in 1995 was 57/1000 total births compared with 11/1000 in high-income countries.[8] Of the estimated seven million perinatal deaths in 1995, more than four million occurred in Asia and over two million in Africa. In settings where many mothers are stunted, do not access antenatal care, and receive poor obstetric care, it seems likely that intrapartum factors remain important in neonatal encephalopathy We aimed to identify avoidable risk factors for neonatal encephalopathy in a low income setting where studies of risk factors have not previously been reported. We focused on neonatal manifestations and maternal factors as the most likely practicalintervention point.

MATERIAL AND METHODS

Pakistan is a low income country with a population of 160 million and an infant mortality rate estimated to be 95/1000 live births in 1994. Hyderabad is the second biggest city of Sindh province. The estimated population of Hyderabad is 02 million.

Newborns with history of delayed cry or Apgar score of less than 7 were admitted. A total of 125 consecutive asphyxiated newborns who met the inclusion criteria were enrolled in the study, with evidence of neurobehavioral disturbance. The findings were recorded on a standard proforma (Table1). A specially designed questionnaire was administered, to assess the role maternal factor and neonatal presentation of asphyxia. Formal operational criteria to grade the severity of encephalopathy in term infants were derived from the syndromic descriptions of Fenichel modified according to more recent studies.[9] In particular, these modifications incorporated the observations that infants with mild neonatal encephalopathy may have signs of not only decreased but increased tone, [10] that seizure activity may not be clinically detectable and therefore cannot serve as a definitive feature in any grading system, [11] and that the inclusion of duration in the clinical definition of a grade[12] renders the scheme contradictory[1]. Infants with mild (Grade 1) encephalopathy were irritable or hyperalert, with either poor suck or an abnormality of tone. Infants with moderate (Grade 2) encephalopathy were lethargic, with moderately abnormal tone, poor suck, and depressed Moro and grasp reflexes (seizures were often clinically evident). Infants with severe (Grade 3) encephalopathy were comatosed, with severely abnormal tone, absent suck, and brainstem malfunction including impaired respiratory drive. Their birth weights and their level of consciousness were also assessed and manifestation was noted as hypoxic ischemic encephalopathy 1, 2 and grade 3. Gestational age less than 34 weeks newborns with major congenital malformations involving central nervous or cardiovascular system, dysmorphism (obvious chromosomal abnormalities), severe hyperbilirubinemia bordering on kernicterus, evidence of meningitis or bleeding disorders were excluded. Maternal characteristics and antepartum history were obtained during an interview of the mothers, using structured proformas. Proforma was filled on each case. Maternal history was taken, regarding their age, gestational age, and complications. Special emphasis was on presentation other then cephalic. Anemia, hypertension, edema and vaginal bleeding was inquired about. Prolonged rupture of membranes, meconium stained liquor and fever was noted. Prolonged rupture labelled when rupture of membranes was more than 18 hours before the birth of baby. Temperature of more than 100°F was considered as febrile. Antenatal care was inquired about in detail. Mode of delivery and instrumentation, if any, was also noted.

In case of clinic or hospital delivery, referral notes were reviewed.

Table 1 - Total no of newborn with birth asphyxia

Presentation	Male	%	Female	%	Total	%
HIE-1	28	22.4	17	13.6	45	36
HIE-2	15	12	10	8	25	20
HIE-3	7	5.6	3	2.4	10	8
No encephalo-pathy	25	20	20	16	45	36
Grand total	75	60	50	40	125	

RESULTS

Among the study population, 75(60%) newborns were males and 50 (40%) were females. 45 (36%) newborns were in Grade I, 25(20%) were in Grade II and 10 (8%) were in grade III. It makes a total of 80(64%) newborns who landed with HIE and 45(36%) newborns having no encephalopathy. 70(56%) presented with poor feeding and 50(40%) newborns were totally unable to feed or suck. Lethargy or inactivity was found in 65(52%), where as irritability was noted in 45(36%). Respiratory difficulty or distress was present in 58(46%). Convulsions were noticed in 35(28%). Decrease tone

was present in 55(44%) where as increased tone was found in 32(25%) of newborns (Table 2).

Table 2 - Clinical presentation of birth asphyxia (n= 125)

Clinical presentation	Number	%
Unable to feed	50	40
Poor feeding	70	56
Cynosis	57	42
Lethargy	65	52
Irritability	45	36
Convulsion	35	28
Respiratory distress	58	46
Decrease tone	55	44
Increased tone	32	25

Table 3 - Maternal factors (n=125)

Factors	Number	%
No antenatal visit	80	64
Antenatal visit to LHV,DAI.other health worker.	25	20
Antenatal to qualified doctor	15	12
Vaginal bleeding	43	34.44
Fever at a time of delivery	30	24
Anemia	75	60
Oedema	57	45.6
Hypertension	24	19.2
Age >35	18	14.4
Age<18	20	16
Age between 18-35	87	69.6
Home delivery	70	56
Delivered at Private clinic OR maternity home	35	28
Delivered at tertiary care hospital	15	12
Mode of delivery NVD	100	80
Mode c-section	25	20
Meconium stained liquor	12	9.6
Prolong rupture of membrane> 18 hours	30	24
Presentation other than cephalic	25	20
Multiple birth	6	4.8

Table 3 lists the intrapartum risk factors. Only 15(12%) mothers had proper antenatal visits to trained medical professionals (doctors or qualified paramedics). 25(20%) visited to untrained birth attendants, or semi-trained midwives, lady health visitor (LHV) or a nurse. Major portion of study population mothers, i.e. 80(64%), did not visit to any medical professional. Most of the mothers, i.e. 70(56%), delivered at home. 35(28%) were delivered at private clinics and maternity homes while only 5(12%) newboms came from tertiary care centers. Place of delivery had significant impact on the grade of encephalopathy In home born newborns, 8(6.4%) had Grade III encephalopathy as compared to the hospital with only 2(1.4%) having Grade III encephalopathy. Around 25(20%) of term infants had a non-cephalic presentation among whom there was a significant excess risk of encephalopathy. A significant risk was associated with prolonged rupture of membranes 30(24%). 25(20%) were delivered by elective caesarean section. Serious intrapartum complications were more common among obstructed labour resulting in a prolonged second stage; normal vaginal delivery was in 100(80%). Maternal hypertension was recorded in 24(19%). Meconium stained liquor and especially thick (particulate) meconium was importantly associated with encephalopathy 12(9.6%). Multiple births were noticed in 6(4.8%) cases. Most of the mothers were between 18-35 years of age. 18(14.4%) were older than 35years, whereas 20(16%) were 18 years and younger. 75(60%) were anemic which is a significant risk factor for asphyxia.

DISCUSSION

Perinatal asphyxia is a global problem causing serious sequelae regarding morbidity and mortality.[13] It is a leading factor contributing in perinatal and neonatal mortality which reflects social, educational and economical standards of a community. Its incidence is very high in developing countries like Pakistan where health facilities are restricted to urban areas and only small population (21%) is getting benefits. Since it is a preventable problem and long-term neurological sequelae are almost untreatable once asphyxia happened, so it is better to avoid at first or if occurred at all, refer as early as possible.

Though the topic has been extensively studied and reviewed worldwide, limited local data is available. When available, the data reflects hospital deliveries, where all kind of resuscitation facilities are available, while almost 80% deliveries are conducted at home in our community where availability of trained personnel and paediatric supervision is lacking[14, 15]. Previously published data proved that there is huge difference in the neonatal deaths among those mothers who had regular antenatal visits as compared to unbooked cases. In a study conducted by Shaheen et al, perinatal mortality rate was 111/1000 live births in non-booked cases as compared to 17/1000 in booked cases.[13] Data from Mayo Hospital, Lahore, Pakistan [16], also stress importance of intervention to reduce the neonatal mortality. In that study, mortality was 27% and again 45% were home-born by untrained persons. Previous published data from the Children Hospital, Lahore, Pakistan, showed that 38% newborns were home-delivered and all of them had HIE while mortality was 40%. In our study 80(64%) had no antenatal visit, which is significant risk for birth asphyxia along with home and untrained delivery practices.

There is no data available in which risk factors are highlighted in asphyxiated newborns. The possible causes of asphyxia could be as simple as hypothermia or hypoglycemia or even diaphragmatic hernia or congenital myopathy, which had to be managed accordingly. So it is important to train people and paramedics about the early recognition and referral of asphyxiated newborns with outcome which depends upon level and duration of neuronal insult at the time of birth, clinical presentations of HIE, involvement of other organs, stage of HIE and treatment modalities.[17, 18] About 10-20% pregnancies are considered as high risk and these include those who have severe cardiac, pulmonary, circulatory problem and diabetes mellitus etc.[19-22]

Despite lot of improvement in the public health over the past many years, it is still a major contributing factor in neonatal mortality.[23] Morbidity and mortality is much higher than it is presented in literature as underreporting is one major factor[24-26]

The impact of long-term neurological sequelae is of utmost importance because it creates tremendous psychosocial and economical burden for the family. This study was hospital based and addressed a common problem of our community Majority of study population as not being delivered under care of trained professionals primarily at home and private clinics also reflects another important issue pertaining to perinatal mortality and morbidity. This preliminary study has shown that antepartum factors may play a role in the etiology of neonatal encephalopathy. . A large proportion of these cases had a significant antepartum history, so that the intrapartum period alone was implicated in the etiology of neonatal encephalopathy. Vaginal bleeding in pregnancy was found to be a significant risk factor for neonatal encephalopathy in this study. An association between antepartum haemorrhage and cerebral palsy has also been shown [19,20]. That early prenatal factors had contributed to the neonatal encephalopathy[30]. Pyrexia during labour and a longer interval between membrane rupture and delivery were associated with neonatal encephalopathy. Fetal sepsis at term has been associated with deterioration in the fetal acid-base status and a prolongation of labour[20, 29] while Nelson and Ellenberg have shown that prolonged rupture of membranes and chorioamnionitis are major predictors of cerebral palsy. The most striking finding relates to mode of delivery. These data suggest an important inverse association between elective caesarean section and newborn encephalopathy[28]. There are several possible explanations for this finding. Chance alone is an unlikely explanation, although mode of delivery was not one of the initial study hypotheses. Anemia is a great risk for asphyxia, probably because of intrapartum hypoxia.

CONCLUSION

Both antepartum and intrapartum factors are important in the causation of neonatal encephalopathy in developing countries. In the short term there are likely to be continuing gains from improvements to intrapartum care. Improvements in the public health of women with associated gains in female growth and nutrition must remain a longer term goal.

Early identification of high-risk cases with improved antenatal and perinatal care can decrease such high mortality. Absent antenatal care was associated with an increased risk of encephalopathy in our study. The safe motherhood policy recommendation of a minimum of three antenatal visits during pregnancy, focusing on risk screening, immunization, anemia prophylaxis and treatment, and health and nutrition education. It is likely that both exposure and outcome are confounded by socioeconomic factors. The only proxy for socioeconomic status we were able to measure was maternal educational status. The effect remains after adjustment for this. It is biologically plausible that risk screening (when combined with appropriate advice and management) and anemia prophylaxis may be protective to the fetus.

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